Pulmonary Tumor Embolism

A Rare Cause of Acute Right Heart Failure with Elevated D-Dimers

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Abstract: We report the case of a 49-year-old woman with a prior history of breast cancer who presented with a subacute course of progressive dyspnoea, culminating in cardiovascular collapse from acute right heart failure. D-dimer serum level was elevated. While a computed tomography of the chest was negative for pulmonary embolism, the autopsy study revealed multiple carcinomatous emboli in distal pulmonary arteries, veins, and lymphatics. Pulmonary tumor embolism may be more frequent than previously thought, and could be mistaken for pulmonary thrombo-embolism.

Key Words: Lung, Embolism, Tumor, Lymphangitis.

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CASE REPORT

The patient was a 49-year-old woman with a history of breast cancer diagnosed 4 years earlier, which was treated by lumpectomy with axillary lymph node resection, adjuvant radiotherapy, and chemotherapy, achieving complete clinical remission. She presented with several weeks of dry cough and exertional dyspnoea. On physical examination, the patient was afebrile, with a respiratory rate of 25/min, a blood pressure of 110/70 mmHg, and a pulse rate of 120/min. There was jugular venous distention, without peripheral edemas. Laboratory results showed thrombocytopenia and an elevated D-dimer serum level (10.000 μg/L, normal $<$500 μg/L). An electrocardiogram demonstrated sinus tachycardia. A chest computed tomography was negative for pulmonary embolism and the only abnormal finding was a slight right upper lobe infiltrate. Transthoracic echocardiography revealed pulmonary hypertension (systolic pressure in the pulmonary artery 70 mmHg) and dilated right heart chambers. The patient's clinical condition worsened, with hemodynamic instability prompting admission to the intensive care unit for hemodynamic support. Pulmonary artery catheterization performed under continuous epinephrin perfusion revealed pulmonary artery pressures of 80/47 mmHg (mean 58 mmHg), a pulmonary capillary wedge pressure of 14 mmHg and a cardiac index of 1.1 L/min/m^2. The patient's clinical condition worsened despite aggressive hemodynamic support and she died 10 hours after admission to the intensive care unit and 1 week after she first consulted her general physician.

A complete autopsy was carried out, with family consent. At macroscopy, lungs displayed bilateral metastases with pleural carcinomatous lymphangitis and dissection of the main arteries showed no thromboemboli. Carinal and peribronchial lymph nodes contained metastatic breast cancer. Right heart hypertrophy (thickness 0.7 cm) with dilatation was seen at heart examination. At histology, the lungs showed carcinomatous thromboemboli in distal pulmonary arteries, veins, and lymphatics, characterized by clusters of cohesive atypical epithelial cells surrounded by an organizing thrombus with and without luminal occlusion (Figure 1) and compatible with a breast origin. Intraparenchymatous pulmonary metastases were observed with glandular differentiation, foci of necrosis and microcalcifications, all typical for an adenocarcinoma. Uninvolved arteries showed concentric medial hypertrophy as typically observed in pulmonary hypertension.

DISCUSSION

Pulmonary tumor emboli are categorized into large, proximal emboli, and smaller emboli in the microvasculature. Although most authors consider pulmonary tumor embolism (PTE) as a rare disease, autopsy series reveal its presence in up to 26% of patients dying from cancer.[1] Malignancies of the breast, stomach, lung, and liver are most commonly associated with PTE.[2] Clinical presentation is generally dominated by subacute and progressive dyspnoea with hypoxemia and tachycardia, but its course may also be acute. PTE and pulmonary thromboembolism are clinically almost indistinguishable, and PTE is often mistaken for thromboembolism. To our knowledge, very high levels of D-dimer have only been rarely reported in the setting of PTE. Although this test is usually used for its high sensitivity at low cutoff values to rule out pulmonary thromboembolism,[3] higher levels are thought to be more specific for this disease and were mis-

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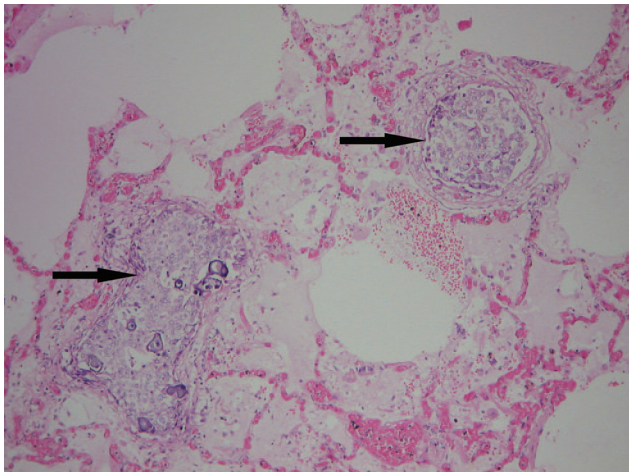

FIGURE 1. Lung histology revealing carcinomatous emboli due to breast cancer (arrows) in the small pulmonary arteries (Haematoxylin and Eosin stain, ×10).

leading in our case. The elevation of D-dimer concentration is most probably explained by activation of the coagulation cascade due to accompanying pulmonary thrombi, and by the underlying neoplastic disease. Chest radiograph and computed tomography are usually unremarkable in PTE, unless there is concomitant carcinomatous lymphangitis. Ventilation-perfusion lung scans may be useful, usually demonstrating multiple distal perfusion defects.[2] Pulmonary microvascular cytology obtained by right heart catherization has been found useful in diagnosing carcinomatous lymphangitis[4]; several authors have also reported its usefulness in PTE without lymphatic involvement. For a definitive diagnosis, surgical open lung biopsy should be obtained. However, aggressiveness for making an accurate diagnosis should be balanced with the almost ineluctably grim outcome of PTE.

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